REMARKS/ARGUMENTS

Claims 1-17 are canceled.

Claims 18-38 are new.

Support for each new claim is found at the originally filed claims and throughout the specification. Additionally, support for reducing is found at paragraph [0006], and is inherent in, for example, Examples 1 and 2. Support for new Claims 37 and 38 is found at page 3, lines 10-16.

Upon entry of the amendment, Claims 18-38 will be active.

No new matter is believed to have been added.

Favorable reconsideration of the claims is requested in light of the arguments and amendments presented in this paper.

The enablement rejection is obviated by cancellation of Claims 1-17.

Applicants have submitted new Claims 18-36.

Claim 18 is drawn to a method of reducing at least one skin damage.

Applicants respectfully submit that Claim 18 is enabled for reducing, and for the listed skin damage conditions.

For example, page 10 of the specification describes that mice fed a feed containing the composition in Claim 18, when compared to mice in a control group, had significantly reduced skin wrinkle formation after being repeatedly subjected to UVB radiation. This example also demonstrates that Claim 18 is enabled for the skin damage: the formation of wrinkles induced by ultraviolet light.

Additionally, page 10 of the specification further describes that the mice fed a feed containing the composition in Claim 18, when compared to the mice in the control group, had significantly reduced formation of verrucae after being exposed to UVB radiation. Only 2 out of 8 of the mice given the feed containing the composition in Claim 18 developed

verrucae, whereas 7 out of 8 of the mice in the control group developed verrucae. Accordingly, Applicants submit Claim 18 is enabled for reducing the formation of verrucae induced by ultraviolet light.

Further, page 12 of the specification describes that mice fed a feed containing the composition in Claim 18, when compared to a control group, developed significantly less erythema than the control group. Accordingly, Applicants submit Claim 18 is enabled for reducing the formation of erythema induced by ultraviolet light.

Regarding enablement for reduction in the formation of freckles and pigmented spots induced by ultraviolet light, Applicants have submitted, along with this paper, an article by Kimura (Exp. Anim. 44(4), 293-229, (1995)).

It is well known in the art that spots and freckles are formed by melanin. UVB (ultraviolet B) exposure, in turn, increases the formation of melanin in the skin. Kimura, for example, at page 297, Figures 7-10, describes marked, moderate, and heavy pigmentation of the epidermis and stratum basale of the skin resulting from exposure to solar radiation (which contains UVA and UVB (Figure 7)) and exposure to UVA +UVB (Figures 8-10). Thus, 1) because UV exposure increases the formation of melanin in the skin, and 2) because freckles and spots are formed from melanin, and 3) because the oral administration of the composition in Claim 18 mitigates the actions of UVB on skin, Applicants submit that the formation of freckles and pigmented spots, caused by the increase in melanin resulting from UVB exposure, would be reduced by the method of Claim 18. Accordingly, Applicants submit Claim 18 is enabled for reducing the formation of freckles and pigmented spots induced by ultraviolet light.

Applicants respectfully submit that Claim 18 is enabled for reducing the formation of skin cancer reduced by ultraviolet light. It is well known in the art that UVB irradiation of skin cells produces DNA lesions including thymine dimmers and photoproducts that are

likely to lead to DNA mutations during replication. UVB radiation is also known to suppress the p53 tumor suppression gene. It is also well accepted in the art that sunscreens, by blocking UV radiation effects on the skin, reduce incidence of skin cancer.

In the Gu reference submitted along with this paper, UVB irradiation in SKH-1 hairless mice was used as a model test for nonmelanoma skin cancer (please see the Abstract of Gu). The experiments of the present specification are identical to the experiment describe in Gu in that 1) animal models were used, 2) skin epidermal damages were induced with UVB radiation, and 3) the intensity of the UVB radiation employed was similar. Because administration of the composition in Claim 18 mitigated the damaging effect of UVB radiation on skin cancer, and because UVB radiation has been used as a model test for nonmelanoma skin cancer, Applicants submit the claims are enabled for reducing the formation of skin cancer reduced by ultraviolet light.

Further, the Alekseev reference submitted along with this paper, describes that when a hairless mouse is irradiated with UVB, squamous cell carcinoma (SCC) subsequently appeared at day 10 post irradiation, and grew larger over time (please see Figures 4C and 4D of Alekseev). Because the present specification describes that skin damage induced by UVB radiation results is reduced by the method of Claim 18, and UVB radiation causes squamous cell carcinoma in an animal model, it is reasonable to conclude that the method of the present claims will reduce the formation of sequmous cell carcinoma in UVB exposed skin. Accordingly, Applicants submit the claims are enabled for reducing the formation of skin cancer reduced by ultraviolet light.

In a similar vein, D'Agnostini describes that UV-B can induce malignant melanoma in hairless mice (please see the Abstract of D'Agnostini). Because the present specification describes that skin damage induced by UVB radiation results is reduced by the method of

Claim 18, and UVB radiation causes malignant melanoma in an animal model, it is reasonable to conclude that the method of the present claims will reduce the formation of malignant melanoma in UVB exposed skin. Accordingly, Applicants submit the claims are enabled for reducing the formation of skin cancer reduced by ultraviolet light.

Additionally, Applicants submit that Claim 18 is enabled for preventing basal cell carcinoma. Figure 1K of Gu describes that UVB-radiation induces DNA-fragmentation in the basal cell layer. Also, Figure 4B of Mittal describes that UVB-irradiation induces DNA methylation in the basal cell layer. The conditions employed by Gu and Mittal are very similar to those employed in the Examples of the present specification, and the Examples of the present specification describe the successful prevention of UVB-irradiation induced skin damage by administering the composition as described in Claim 18. Accordingly, Applicants submit the present specification is enabled for preventing basal cell carcinoma.

Finally, Applicants submit the substituents described in, for example, formula (I) of Claim 18, are enabled, in that one of ordinary skill in the art would readily be able to ascertain, for example, the compounds which belong to: C_{12-24} aliphatic hydrocarbon group having a degree of unsaturation of between 0 and 2. Applicants note, for the Office's convenience, that Claims 20 and 21 have been added, which describe specific examples of substituents for the compound of formula (I).

Applicants submit the present application is now in condition for allowance. Early notification to this effect is earnestly solicited.

Respectfully submitted,

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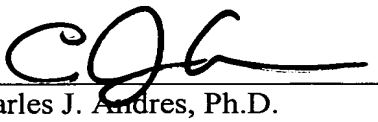

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